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Term: L23 and protein

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DATE: Tuesday, April 26, 2005 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT; PLUR=YES; OP=ADJ		
L27	L25 and protein same embeded	0	L27
L26	L25 and protein same embedded	0	L26
L25	L23 and protein	63	L25
L24	containing same protein	70959	L24
L23	210/500.35.ccls.	168	L23
L22	enrgy converting proteins and membrane and polymer	0	L22
L21	enrgy converting proteins and membrane and polymer and vinyl	0	L21
L20	membrane and cytochrome oxydase	2	L20
L19	copolymer same cytochrome oxydase	0	L19
L18	L15 and cytochrome oxydase	0	L18
L17	L15 and bacteriodopsin	0	L17
L16	L15 and adding protein	0	L16
L15	l5 and copolymer and protein	75	L15
L14	l5 and adding protein	0	L14
L13	l5 and adding protein same membrane	0	L13
L12	membrane same aquaporin	41	L12

EAST - [Untitled1:1]

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- Drafts
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  - L1: (10) (("6319469") or ("6342389") or ("6355423") or ("6329209") or ("61
  - L2: (8) (("6319459") or ("6342389") or ("6355423") or ("6329209")).PN.
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	U	1	Document ID	Issue Date	Pages	Title	Current OR	Current XRef	Retrieval Cla	Inventor	S	C	P	2	3	4	5	Int
1	☐	☐	US 6355423 B1	20020312	78	Methods and devices for measuring differential gene expression	435/6	435/91.2; 536/23.1;		Rothberg; Jonathan Marc et al.	☑	☐	☐	☐	☐	☐	☐	US
2	☐	☐	US 6342389 B1	20020129	29	Modified phycobilisomes and uses therefore	435/317.1	435/4; 435/7.1;		Cubicciotti; Roger S.	☑	☐	☐	☐	☐	☐	☐	US
3	☐	☐	US 6329209 B1	20011211	34	Arrays of protein-capture agents and methods of use thereof	436/518	427/261; 435/287.9		Wagner; Peter et al.	☑	☐	☐	☐	☐	☐	☐	US
4	☐	☐	US 6319459 B1	20011120	4	Removal of organic acid based binders from powder metallurgy com	419/26	148/513; 419/28;		Melody; Brian John et al.	☑	☐	☐	☐	☐	☐	☐	US
5	☐	☐	US 6342389 B	20020129	29	New modified or isolated phycobilisomes, or phycobilisome co				CUBICCIOTTI, R S	☑	☐	☐	☐	☐	☐	☐	US
6	☐	☐	US 6329209 B	20011211	34	Array of protein-capture agents useful for proteomics and assaying differenti				AULT-RICHE, D et al.	☑	☐	☐	☐	☐	☐	☐	US
7	☐	☐	US 6319459 B	20011120	4	Removal of organic acid-based binders from a powder metallurgy co				KINARD, J T et al.	☑	☐	☐	☐	☐	☐	☐	US
8	☐	☐	US 6355423 B	20020312	78	Identifying and quantitating nucleic acids				HU, X et al.	☑	☐	☐	☐	☐	☐	☐	US

Ready NUM

L11	aquaporin same polymer same membrane membrane	0	L11
L10	membrane and l5 and aquaporin	0	L10
L9	membrane ontaining protein	0	L9
L8	membrane ontaining protein	0	L8
L7	L6 and copolymer	17	L7
L6	L5 and protein same containing	24	L6
L5	210/500.42.ccls.	346	L5
L4	L3 and immobilized	10	L4
L3	L2 and l1	47	L3
L2	210/500.27.ccls.	394	L2
L1	membrane and protein and pores and filter	10661	L1

END OF SEARCH HISTORY

L2: Entry 3 of 19 File: USPT Dec 21, 2004

DOCUMENT-IDENTIFIER: US 6833438 B1
TITLE: Serpentine transmembrane antigens expressed in human cancers and uses thereof

Abstract Text (1):
Described is a novel family of cell surface serpentine transmembrane antigens. Two of the proteins in this family are exclusively or predominantly expressed in the prostate, as well as in prostate cancer, and thus members of this family have been termed "STEAP" (Six Transmembrane Epithelial Antigen of the Prostate). Four particular human STEAPs are described and characterized herein. The human STEAPs exhibit a high degree of structural conservation among them but show no significant structural homology to any known human proteins. The prototype member of the STEAP family, STEAP-1, appears to be a type IIIa membrane protein expressed predominantly in prostate cells in normal human tissues. Structurally, STEAP-1 is a 339 amino acid protein characterized by a molecular topology of six transmembrane domains and intracellular N- and C-termini, suggesting that it folds in a "serpentine" manner into three extracellular and two intracellular loops. STEAP-1 protein expression is maintained at high levels across various stages of prostate cancer. Moreover, STEAP-1 is highly over-xpressed in certain other human cancers.

Brief Summary Text (14):
There are some known markers which are expressed predominantly in prostate, such as prostate specific membrane antigen (PSM), a hydrolase with 85% identity to a rat neuropeptidase (Carter et al., 1996, Proc. Natl. Acad. Sci. USA 93: 749; Bzdega et al., 1997, J. Neurochem. 69: 2270). However, the expression of PSM in small intestine and brain (Israeli et al., 1994, Cancer Res. 54: 1807), as well its potential role in neuropeptide catabolism in brain, raises concern of potential neurotoxicity with anti-PSM therapies. Preliminary results using an indium-111 labeled, anti-PSM monoclonal antibody to image recurrent prostate cancer show some promise (Sodee et al., 1996, Clin Nuc Med 21: 759-766). More recently identified prostate cancer markers include PCTA-1 (Su et al., 1996, Proc. Natl. Acad. Sci. USA 93: 7252) and prostate stem cell antigen (PSCA) (Reiter et al., 1998, Proc. Natl. Acad. Sci. USA 95: 1735). PCTA-1, a novel galectin, is largely secreted into the media of expressing cells and may hold promise as a diagnostic serum marker for prostate cancer (Su et al., 1996). PSCA, a GPI-linked cell surface molecule, was cloned from LAPC-4 cDNA and is unique in that it is expressed primarily in basal cells of normal prostate tissue and in cancer epithelia (Reiter et al., 1998). Vaccines for prostate cancer are also being actively explored with a variety of antigens, including PSM and PSA.

Brief Summary Text (17):
The prototype member of the STEAP family, STEAP-1, appears to be a type IIIa membrane protein expressed predominantly in prostate cells in normal human tissues. Structurally, STEAP-1 is a 339 amino acid protein characterized by a molecular topology of six transmembrane domains and intracellular N- and C- termini, suggesting that it folds in a "serpentine" manner into three extracellular and two intracellular loops. STEAP-1 protein expression is maintained at high levels across various stages of prostate cancer. Moreover, STEAP-1 is highly over-expressed in certain other human cancers. In particular, cell surface expression of STEAP-1 has been definitively confirmed in a variety of prostate and prostate cancer cells,

bladder cancer cells and colon cancer cells. These characteristics indicate that STEAP-1 is a specific cell-surface tumor antigen expressed at high levels in prostate, bladder, colon, and other cancers.

Drawing Description Text (8):
FIG. 7. Cell surface biotinylation of STEAP-1. FIG. 7A: Cell surface blotinylation of 293T cells transfected with vector alone or with vector containing cDNA encoding 6His-tagged STEAP-1. Cell lysates were immunopredpitated with specific antibodies, transferred to a membrane and probed with horseradish peroxidase-conjugated streptavidin. Lanes 1-4 and 6 correspond to immunopredpitated from lysates prepared from STEAP-1 expressing 293T cells. Lanes 5 and 7 are immunoprecipitates from vector transfected cells. The immunoprecitations were performed using the following antibodies: (1) sheep non-immune, (2) anti-Large T antigen, (3) anti-CD71 (transferrin receptor), (4) anti-his, (5) anti-His, (6) anti-STEAP-1, (7) anti-STEAP-1. FIG. 7B: Prostate cancer (LNCaP, PC-3, DU145), bladder cancer (UM-UC-3, TCCSUP) and colon cancer (LOVO, COLO) cell lines were either biotinylated (+) or not (-) prior to lysis. Western blots of streptavidin-gel purified proteins were probed with anti-STEAP-1 antibodies. Molecular weight markers are indicated in kilodaltons (kD).

Detailed Description Text (6):
As used herein, the term "polypeptide" means a polymer of at least 8 amino acids. Throughout the specification, standard three letter or single letter designations for amino acids are used.

Detailed Description Text (12):
The prototype member of the STEAP family, STEAP-1, is a six-transmembrane cell surface protein of 339 amino acids with no identifiable homology to any known human protein. The cDNA nucleotide and deduced amino acid sequences of human STEAP-1 are shown in FIG. 1A. A gross topological schematic of the STEAP-1 protein integrated within the cell membrane is shown in FIG. 1B. STEAP-1 expression is predominantly prostate-specific in normal tissues. Specifically, extensive analysis of STEAP-1 mRNA and protein expression in normal human tissues shows that STEAP-1 protein is predominantly expressed in prostate and, to a far smaller degree, in bladder. STEAP-1 mRNA is also relatively prostate specific, with only very low level expression detected in a few other normal tissues. In cancer, STEAP-1 mRNA and protein is consistently expressed at high levels in prostate cancer (including androgen-dependent and androgen-independent tumors) and during all stages of the disease. STEAP-1 is also expressed in other cancers. Specifically, STEAP-1 mRNA is expressed at very high levels in bladder, colon, pancreatic, and ovarian cancer (as well as other cancers). In addition, cell surface expression of STEAP-1 protein has been established in prostate, bladder and colon cancers. Therefore, STEAP-1 has all of the hallmark characteristics of an excellent therapeutic target for the treatment of certain cancers, including particularly prostate, colon and bladder carcinomas.

Detailed Description Text (17):
The function of the S are not known. Other cell surface molecules that contain six transmembrane domains include ion channels (Dolly and Parcel, 1996 J Bioenerg Blomembr 28:231) and water channels or aquaporins (Reizer et al., 1993 Crit Rev Biochemn Mol Biol 35 28:235). Structural studies show that both types of molecules assemble into tetrameric complexes to form functional channels (Christie, 1995, Clin Exp Pharmacol Physiol 22:944, Walz et al., 1997 Nature 387:624, Cheng et al., 1997 Nature 387:627). Immunohistochemical staining of STEAP-1 in the prostate gland seems to be concentrated at the cell-cell boundaries, with less staining detected at the luminal side. This may suggest a role for STEAP-1 in tight-junctions, gap-junctions or cell communication and adhesion. In order to test these possibilities, xenopus oocybes (or other cells) expressing STEAP may be analyzed using voltage-clamp and patch-clamp experiments to determine if STEAP functions as an ion-channel. Oocyte cell volume may also be measured to determine if STEAP exhibits

water channel properties. If STEAPs function as channel or gap-junction proteins, they may serve as excellent targets for inhibition using, for example, antibodies, small molecules, and polynucleotides capable of inhibiting expression or function. The restricted expression pattern in normal tissue, and the high levels of expression in cancer tissue suggest that interfering with STEAP function may selectively kill cancer cells.

Detailed Description Text (101):
Various ex vivo strategies may also be employed. One approach involves the use of dendritic cells to present STEAP antigen to a patient's immune system. Dendritic cells express MHC class I and II, B7 costimulator, and IL-12, and are thus highly specialized antigen presenting cells. In prostate cancer, autologous dendritic cells pulsed with peptides of the prostate-specific membrane antigen (PSMA) are being used in a Phase I clinical trial to stimulate prostate cancer patients' immune systems (Tjoa et al., 1996, Prostate 28: 65-69; Murphy et al., 1996, Prostate 29: 371-380). Dendritic cells can be used to present STEAP peptides to T cells in the context of MHC class I and II molecules. In one embodiment, autologous dendritic cells are pulsed with STEAP peptides capable of binding to MHC molecules. In another embodiment, dendritic cells are pulsed with the complete STEAP protein. Yet another embodiment involves engineering the overexpression of the STEAP gene in dendritic cells using various implementing vectors known in the art, such as adenovirus (Arthur et al., 1997, Cancer Gene Ther. 4: 17-25), retrovirus (Henderson et al., 1996, Cancer Res. 56: 3763-3770), lentivirus, adeno-associated virus, DNA transfection (Ribas et al., 1997, Cancer Res. 57: 2865-2869), and tumor-derived RNA transfection (Ashley et al., 1997, J. Exp. Med. 186: 1177-1182).

Detailed Description Text (176):
The results are shown in FIG. 8. LNCaP cells showed uniformly strong peri-cellular staining in all cells (FIG. 8b). Excess STEAP N-terminal peptide (peptide 1) was able to competitively inhibit antibody staining (FIG. 8a), while peptide 2 had no effect (FIG. 8b). Similarly, uniformly strong peri-cellular staining was seen in the LAPC-9 (FIG. 8f) and LAPC4 prostate cancer xenografts (data not shown). These results are dear and suggest that the staining is STEAP specific. Moreover, these results visually localize STEAP to the plasma membrane, corroborating the biochemical findings presented in Example 4 below.

Detailed Description Text (177):
The results obtained with the various clinical specimens are show in FIG. 8c (normal prostate tissue), FIG. 8d (grade 3 prostatic carcinoma), and FIG. Be (grade 4 prostatic cardnoma), and are also included in the summarized results shown in TABLE 1. Light to strong staining was observed in the glandular epithelia of all prostate cancer samples tested as well as in all samples derived from normal prostate or benign disease. The signal appears to be strongest at the cell membrane of the epithelial cells, especially at the cell-cell junctions (FIG. 8c, d and e) and is also inhibited with excess STEAP N-terminal peptide 1 (data not shown). Some basal cell staining is also seen in normal prostate (FIG. 8c), which is more apparent when examining atrophic glands (data not shown). STEAP-1 seems to be expressed at all stages of prostate cancer since lower grades (FIG. 8d), higher grades (FIG. 8e) and metastatic prostate cancer (represented by LAPC-9; FIG. 8f) all exhibit strong staining.

Detailed Description Text (181):
To initially characterize the STEAP-1 protein, cDNA clone 10 was cloned into the pcDNA 3.1 Myc-His plasmid (Invitrogen), which encodes a 6His tag at the carboxyl-terminus, transfected into 293T cells, and analyzed by flow cytometry using anti-His monocIonal antibody (His-probe, Santa Cruz) as well as the anti-STEAP-1 polycIonal antibody described above. Staining of cells was performed on intact cells as well as permeabilized cells. The results indicated that only permeabilized cells stained with both antibodies, suggesting that both termini of the STEAP-1 protein are localized intracellularly. It is therefore possible that one or more of

the STEAP-1 protein termini are associated with intracellular organelles rather than the plasma membrane.

Detailed Description Text (182):
To determine whether STEAP-1 protein is expressed at the cell surface, intact STEAP-1-transfected 293T cells were labeled with a biotinylation reagent that does not enter live cells. STEAP-1 was then immunoprecipitated from cell extracts using the anti-His and anti-STEAP antibodies. SV40 large T antigen, an intracellular protein that is expressed at high levels in 293T cells, and the endogenous cell surface transferrin receptor were immunoprecipitated as negative and positive controls, respectively. After immunoprecipitation, the proteins were transferred to a membrane and visualized with horseradish peroxidase-conjugated streptavidin. The results of this analysis are shown in FIG. 7. Only the transferrin receptor (positive control) and STEAP-1 were labeled with biotin, while the SV40 large T antigen (negative control) was not detectably labeled (FIG. 7A). Since only cell surface proteins are labeled with this technique, it is clear from these results that STEAP-1 is a cell surface protein. Combined with the results obtained from the flow cytometric analysis, it is clear that STEAP-1 is a cell surface protein with intracellular amino- and carboxyl-termini.

Detailed Description Text (183):
Furthermore, the above results together with the STEAP-1 secondary structural predictions, shows that STEAP-1 is a type IIIa membrane protein with a molecular topology of six potential transmembrane dornains, 3 extracellular loops, 2 intracellular loops and two intracellular termini. A schematic representation of STEAP-1 protein topology relative to the cell membrane is shown in FIG. 1B.

Other Reference Publication (19):
A. Lepple-Wienhues et al. (1996) "K+ Channels and the Intracellular Calcium Signal in Human Melanoma Cell Proliferation," J. Membrane Biol. 151:149-157.

Other Reference Publication (20):
A.A. Marino et al. (1994) "Association between Cell Membrane Potential and Breast Cancer," Tumor Biol. 15:82-89.

Previous Doc Next Doc Go to Doc#